psychologically structured interviews, has been well documented. Measuring the serum prolactin level immediately following an attack has been advocated as a diagnostic aid and, when elevated, may prove useful in confirming epileptic seizures.

There is no widely accepted effective treatment for pseudoseizures. There has been a renewed interest in the use of hypnosis, but its effectiveness in this disorder must be carefully evaluated in a controlled fashion. Therefore, an accurate diagnosis remains the single most important step in managing pseudoseizures.

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Should Hypertension in a Patient With Acute Stroke Be Treated?

COMMON MEDICAL WISDOM, still widely taught and believed in this country, holds that elevated blood pressure should be lowered during ischemic stroke and certain other cerebrovascular disorders. Although this dogma has been questioned in recent years, most practicing physicians still use antihypertensive measures immediately after cerebral infarction and in patients with subarachnoid or intracerebral hemorrhage.

High blood pressure is very common early in the course of acute stroke, being found in 70% or more of these cases when seen initially. Of those patients not treated for hypertension, the great majority become normotensive within ten days. The blood pressure rise associated with the onset of cerebral ischemia apparently subsides as brain function recovers and autoregulation is restored. In such persons a sudden new blood pressure elevation may suggest a recurrence of brain ischemia. Bilateral carotid occlusion in animals is accompanied by an immediate rise in the blood pressure, which generally is proportional to the fall in cerebral perfusion. These observations indicate that hypertension in a patient with acute stroke often is a physiologic mechanism designed to protect ischemic brain by increasing the blood flow.

Cerebral autoregulation is impaired in blood vessels irrigating that part of the brain where the stroke has occurred. Thus, blood flow to this area becomes passively dependent on arterial pressure. If the blood pressure falls, perfusion of the ischemic area declines, thereby leading to a possible increase of the infarct and jeopardizing recovery. Decreased flow also may encourage further thrombus formation and could cause additional narrowing in the originally affected artery.

Many physicians believe that hypertension worsens the outcome of acute cerebral infarction, perhaps by causing forced vasodilation, bleeding and cerebral edema. There is little clinical evidence to support this thesis. On the contrary, data exist to indicate that hypotension during an acute stroke, either through medication or from other mechanisms, may worsen rather than improve the clinical outcome by extending the original infarct and impairing flow through the watershed area.

There is some uncertainty as to the specific indications for treating hypertension in patients with acute stroke. Some experts feel that blood pressures elevated to levels of 220/130 mm of mercury or more always should be treated. Most experts recommend that therapy for high blood pressure is essential in the face of myocardial insufficiency (especially congestive heart failure), severe renal damage, retinal bleeding and when hypertensive encephalopathy clearly is present. Hypertensive encephalopathy, a term coined by Oppenheimer and Fishberg in 1928 for recurrent seizures in patients with acute nephritis with elevated blood pressure, is uncommon, is probably due to a breakdown of cerebral autoregulation and is characterized by weakness, apathy, headaches, drowsiness, seizures, visual loss, transient and often fluctuating focal neurologic signs plus coma.

As regards subarachnoid hemorrhage, many authorities now believe that induced hypertension along with volume expansion is useful to counter vasospasm. There is no clear-cut evidence that reducing the blood pressure in patients with hypertensive intracerebral hemorrhage is or is not helpful, and some literature indicates that a high blood pressure should not be lowered in this situation.

The above discussion relates only to acute stroke. The fact that chronic hypertension is the most common and potent risk factor for cerebral infarction remains undisputed. There also is good evidence that treating hypertension reduces the occurrence of stroke, and there are strong indications in the literature that lowering blood pressure after a cerebral infarction reduces the risk for subsequent events.

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Panic Disorder

ONE OF THE more interesting and rapidly changing neuropsychiatric conditions involves a heterogeneous group of disorders currently subsumed under the general heading "Panic Disorder." Although defined in the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition (DSM III), as "agoraphobia, with or without panic attacks," many clinicians find this classification inadequate. It is anticipated that the revised edition of DSM III (DSM III-R) will be a more useful reference. This topic was reviewed in the March 1983 issue of this journal and interested readers are referred to it.

Symptoms have been described as being due to a "profound activation of the sympathetic nervous system." They are primarily cardiovascular and respiratory in nature: dyspnea, palpitations, chest pain or discomfort, choking or smothering sensations, or a more general reflection of autonomic nervous system dysfunction—that is, dizziness; feelings of unreality; paresthesias in hands or feet; hot and cold flashes; sweating; faintness; trembling or shaking; fear of dying, "going crazy" or doing something uncontrolled during an attack, or a sense of apprehension or impending doom. While usually lasting for only minutes, they may persist for hours. The symptoms are accompanied by varying degrees of

nervousness and apprehension between attacks. As one might readily predict, afflicted patients may initially be seen by any of a group of specialists—a cardiologist, neurologist, gastroenterologist, internist or an emergency department physician. As more detailed physical examinations usually reveal little, the patient is reassured and told not to worry. After several episodes similar to this, both physician and patient feel frustrated, the patient often feeling abused and angry and sometimes even frightened. The idea of seeing a psychiatrist (sort of a medical "court of last resort") may be brought up—an often serendipitous choice.

Further clinical evaluation often reveals that, while initial anxiety attacks may have been spontaneous, most patients quickly discover those situations most likely to induce symptoms and begin to structure their lives to minimize their occurrence. Often, elaborate "hierarchies" of "safe" and "not-safe" activities and places are the result, leading to greatly disrupted family and personal lives.

Biologic factors are becoming increasingly important. Although data are often minimal and inconsistent, the following trends have appeared:

- Cross-cultural prevalence is between 1% and 4%, with women twice as likely to be afflicted as men.
- There is a higher concordance rate in monozygotic twins than in dizygotic twins and nontwin siblings.
- Patients with panic disorder are at increased risk for a depressive disorder.
- There is a higher rate of alcoholism, panic disorder and depression in probands of patients with this disorder.
- There is a uniform, unimodal age of onset distribution in the early 20s among afflicted patients.
- Abnormal sleep patterns, atypical responses to sleep deprivation, blunted growth hormone response to administration of clonidine hydrochloride and blunted thyroid-stimulating hormone and prolactin responses to thyrotropin-releasing hormone have been documented.
- Mitral valve prolapse occurs in up to 50% of patients with panic disorder. As many patients are also tall, thin, with long arms and narrow anteroposterior chest diameters, a systemic disorder marked by skeletal abnormalities, mitral valve prolapse and autonomic nervous system dysfunction has been postulated.
- About 80% of these patients have a panic attack when given a lactate infusion test as compared with only moderate and transient symptoms in a much smaller percentage of normal persons.
- Both the locus ceruleus and the left parahippocampal gyrus (the latter by positron emission tomography) have been found to be abnormal in many patients with panic attacks.

Treatment for the condition is divided into several types—each with its strong proponents ranging from an almost purely in vivo behavioral desensitization program to a more classical psychodynamic approach. Of the medications used, the tricyclic antidepressant imipramine, the new second-generation azolobenzodiazepine, alprazolam, and the monoamine-oxidase inhibitor phenelzine are the mainstays. A middle-of-the-road therapeutic approach involves these medications, together with behavioral exposure and cognitive and supportive psychological adjuncts. While 90% to 95% of patients are successfully treated, relapse rates range from 30% to 75%. Several courses of treatment are often necessary.

In conclusion, while theories of causation have been suggested along biologic and psychological lines, none are wholly consistent with the findings regarding the phenomenology and evolution of panic disorder. It would be premature to conclude that any unitary conceptual framework has yet been provided, and the biologic, psychological and behavioral aspects of this disorder should all receive due attention both in clinical management and scientific investigation.

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Current Concepts—Alcohol and Brain Damage

FOR MANY YEARS central nervous system dysfunction associated with chronic alcoholism was attributed to trauma, infection, liver disease and, especially, nutritional deficiency rather than to a direct toxic effect of alcohol. The Wernicke-Korsakoff syndrome, a serious and well-known complication of long-standing alcohol ingestion, was blamed on thiamine deficiency. The average alcoholic person was felt not at risk for cerebral damage if nutrition was maintained. Newer data from animal studies, neuropsychological tests and computed tomography suggest that alcohol indeed is directly injurious to the central nervous system regardless of food intake.

There is evidence that in 50% or more of those with chronic alcoholism, cognitive changes develop that are characterized by a radiologically demonstrable, probably reversible, cortical atrophy and specific neuropsychological deficits. Among the neuropsychological deficits are a progressive disinterest in the environment, carelessness about personal appearance, impaired judgment, defective intention, slowness of thought and, on specific testing, visuoperceptive and problem-solving abnormalities. This condition, termed alcoholic dementia, is attributed to the direct effects of alcohol on the association cortex, occurs even with good nutrition and is said to depend on the duration, amount and frequency of ethanol consumption. These patients usually have a mild but not readily apparent memory loss.

In contrast, those with the Korsakoff syndrome have an anterograde amnesia manifested by interference with the transfer of information from short- to long-term memory and also a retrograde amnesia with impaired retrieval of stored information. The latter has a temporal gradient with more difficulty in recalling events close to the onset of illness than those of a more remote nature. These patients usually have relatively intact intelligence by a standard IQ test, although their memory quotient is defective. They also share the specific neuropsychological deficits of the type noted above in alcoholic dementia.

Thus, little doubt now exists that most persons with chronic alcoholism have cognitive deficits that often are mild and detectable only through relatively advanced testing. These losses appear to be of cortical origin, can be accompanied by reversible cortical atrophy and seem unrelated to nutrition. On the other hand, Korsakoff's syndrome is a se-